No. 1318

Experimental and Theoretical Analysis of Cochlear K^+-Circulation in the Inner Ear

Fumiaki Nin, Takamasa Yoshida, Hiroshi Hibino

Department of Molecular Physiology, Niigata University

Summary

The endocochlear potential (EP) of +80 mV in the scala media, which is indispensable for audition, is controlled by K^+ transport across the lateral cochlear wall. This wall includes two epithelial barriers, the syncytium and the marginal cells. The former contains multiple cell types, such as fibrocytes, which are exposed to perilymph on their basolateral surfaces. The apical surfaces of the marginal cells face endolymph. Between the two barriers lies the intrastrial space (IS), an extracellular space with a low K^+ concentration ($[K^+]$) and a potential similar to the EP. This intrastrial potential (ISP) dominates the EP and represents the sum of the diffusion potential elicited by a large K^+ gradient across the apical surface of the syncytium and the syncytium's potential, which is slightly positive relative to perilymph. Although a K^+ transport system in fibrocytes seems to contribute to the EP, the mechanism remains uncertain. We examined the electrochemical properties of the lateral wall of guinea pigs with electrodes sensitive to potential and K^+ while perfusing into the perilymph of the scala tympani blockers of Na^+, K^+-ATPase and Na^+, K^+, 2Cl- cotransporter (NKCC) the K^+ pump thought to be essential to the system. Inhibiting Na^+, K^+-ATPase barely affected $[K^+]$ in the IS but greatly decreased $[K^+]$ within the syncytium, reducing the K^+ gradient across its apical surface. The treatment hyperpolarized the syncytium only moderately. Blocking NKCCs by perilymphatic perfusion of bumetanide suppressed the ISP. Unexpectedly and unlike the inhibition of the syncytial Na^+, K^+-ATPases, the bumetanide perfusion barely altered the electrochemical properties of the syncytium but markedly augmented $[K^+]$ of the IS. These observations resembled those when the marginal-cells' Na^+, K^+-ATPases or NKCCs were blocked with vascularly applied inhibitors. Consequently, fibrocytes evidently use the Na^+, K^+-ATPase to achieve local K^+ transport, maintaining the syncytium's high $[K^+]$ that is crucial for the K^+ diffusion underlying the positive ISP. To the contrary, NKCCs in the marginal cells are affected by the perilymphatically perfused bumetanide and these transporters but not those in the syncytium are involved in the unidirectional K^+-transport.